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WITH ABSTRACT OF THE LITERATURE

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The Infectivity of Silicotic Tuberculosis

The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers
(*Concluded*)

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THE JOURNAL OF INDUSTRIAL HYGIENE

VOLUME XII JUNE, 1930 NUMBER 6

PERSONAL QUALITIES IN ACCIDENT CAUSATION*

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THE main purpose of this article is to give some account of the practical and theoretical difficulties encountered in a research into the personal qualities involved in accident causation. This problem is exceedingly important and is, in a sense, one of the main problems of psychology since its solution demands a stringent research into mental integration. The occurrence of an accident, as I shall show later, is not necessarily fortuitous but is often the result of the complicated interaction of various psychologic and physiologic functions; and a research which has as its object the disentanglement of such complex mental situations (using mental in its widest sense) is bound to throw light on the nature of mental life as a whole. The accident situation is one in which everybody finds himself sooner or later, and to which he reacts either by incurring an accident or by avoiding one. If we can find out why some people react in the one way and some in the other, we shall have learned a good deal about the nature of the reactions of mind to its environment.

About the practical importance of this problem to industry there can be little doubt. The cost of accidents is enormous not only in actual money but also in loss of time and efficiency. It is difficult to gauge the full extent of this latter loss, but the financial cost of accidents, as represented by the annual expenditure in insurance premiums, runs into hundreds of thousands of pounds. This insurance, of course, only covers cases where compensation has to be paid, and compensated accidents are actually only 1 or 2 per cent. of the total number of industrial accidents.

An accident is commonly regarded as being due to chance: that is, as an event the causes of which are external to the sufferer and not affected in any

* Received for publication March 26, 1930.

way by the nature of the sufferer himself. If this were so, one person would be just as likely to sustain an accident as would another in circumstances of equal risk; and if records were taken for a large enough group of workers over a sufficient period of equal exposure to risk, it would be found that the accidents sustained during that period were equally distributed among the people concerned. In 1919 Greenwood and Woods (1) showed that the curve of the frequency distribution of accidents is not a straight line or a normal, saddleback curve, but is a curve shaped rather like the letter J. This was confirmed by Newbold (3) in 1926. Broadly interpreted, this fact means that some people have more and some fewer accidents than can be accounted for by the operation of chance alone—a fact of great importance from a psychologic standpoint since it definitely postulates the existence of a liability to have accidents which is more or less constant for each person but varies from one person to another. In other words, accidents are due not solely to chance but also to some personal quality which we may call accident proneness. This fact alone gives a *prima facie* warrant for a study of individual psychologic differences; it was the basis of the subsequent practical work done by the Industrial Health Research Board[1] on the causation of accidents.

Another fact of practical importance which may be deduced from the J-shaped distribution curve of accidents is that approximately 70 per cent. of the accidents sustained by a group of workers are actually sustained by only 25 per cent. of the workers; therefore, by weeding out a quarter of the workers a very considerable reduction in accident incidence would be effected.

All the accident records which have yet been examined yield the J-shaped distribution curve, and all agree with the theory that the individuals in the groups concerned differ in their proneness to accidents. Newbold (3) has shown that the frequency distribution curve for foreign bodies in the eye—a particular type of accident—is likewise J-shaped, so that even in this apparently unlikely instance the theory of differing personal proneness is confirmed.

Types of Accidents

Accidents may be divided roughly into three categories. First there are those which are purely accidental in the popular sense of the word; in the second class are accidents which depend on the instrumentality of other people and not on the sufferer himself; and third there are the accidents which depend directly on one's individual proneness. The first of these divisions comprises what insurance policies call "Acts of God," which consist essentially in such injuries to the person as are the inevitable results of nonhuman environmental changes. The second class of accidents is similar to the first from the point of view of the sufferer. Again environmental change results in inevitable injury to him, but in this case the change is produced by human agency. Finally, there is the class which embraces accidents that are the result of environmental changes either produced by the sufferer himself or of such

[1] Throughout the text the Board is referred to uniformly under its new name; in the bibliography the name used is that given on the cover of the report.

a nature as to be avoidable by certain reactions on his part.

The point of this classification is that the first and second categories are marked by the inevitability of the accident: the sufferer cannot escape the accident whether the causes are human or nonhuman—he may be struck by lightning or he may be shot by a careless sportsman. Any accidents which are directly avoidable properly fall into the third category, and it is only accidents in this class which can be regarded as due to personal proneness. As yet there is no evidence to show what proportion of accidents fall into each of these classes, but the universality of the J-shaped distribution curve shows definitely that the majority of industrial accidents belong to the third category: that is, that they are due to personal proneness. There is, of course, a certain amount of overlap between these classes. For example, the activity of an accident-prone worker may produce an accident which is sustained not by himself but by another worker near him. Really such an accident should count as a third-category accident to the first worker; actually in the records it would figure as a second-category accident to the worker who sustained it.

It will be seen at the outset that any research into accident causation is handicapped by uncertainty as to the type of accidents sustained by any group of workers. It is also obvious that no investigation can lead to changes in practice which would completely eliminate accidents, since accidents in the first and second categories are not due to personal differences.

The foregoing remarks suggest a definition for the accidents which are due to personal proneness. Such an accident may be defined as a physical injury due to a faulty reaction on the part of the sufferer to some environmental change. The causes of this faulty reaction lie in the sufferer himself, and it is the attempt to discover the nature of these causes which constitutes the research which Mr. Eric Farmer and I are making in this field (4) (5).

Difficulties in Practical Research

So far, for the sake of convenience in argument, accident proneness has been alluded to as a personal *quality*, but it is by no means necessarily the case that such proneness is a *single* quality. A study of the nature of accidents themselves suggests, on the contrary, that this proneness is complex in nature and the resultant of a number of psychologic and physiologic functions. For example, speed of movement, muscular control, and general intelligence, all seem to play a part in the avoidance of such accidents as cut or crushed fingers and burns. Further, there is nothing to indicate that these functions are combined in certain definite proportions to produce a new, integral function which we have called accident proneness; rather it would seem that in some cases one function predominates, and in other cases another. Hence, proneness may be regarded as a group of co-operating functions and not as a definite unitary function, and this in its turn forms a further handicap to practical research in this field. Not only the type of

accident may vary, but also the causes of the same type of accident may vary.

Another difficulty arises immediately we begin to consider the measurement of accident proneness, for it is evident that proneness and number of accidents sustained are not synonymous. We cannot say that a person with a high degree of accident proneness will have many accidents and that another person with a low degree of proneness will have few accidents. Such a statement would leave entirely out of account all accidents falling into the first two categories and also inequalities of exposure to risk. All we can definitely say is that in a group of people equally exposed to risk but of varying accident proneness, those with the greater proneness to accidents will tend to sustain a larger number of accidents than those with a lesser proneness. Of course, the longer the period over which accidents are recorded, the nearer does the actual accident rate approximate a true measure of accident proneness; therefore, the accident rate of the whole working life would be the closest measure of accident proneness that is possible.

Some of the practical difficulties connected with the collection of accident records may be mentioned here. These records are not always kept in a way which makes analysis easy. This is to be expected since the records are usually kept to serve practical purposes and not as a basis for statistical inquiry. In most cases a bare statement of the occurrence of an accident to a particular person on a particular date is all that is recorded. The nature of the accident is sometimes given, but its gravity is rarely recorded. Moreover it is sometimes difficult to distinguish between redressings of the same wound and further accidents. Apart from this there is rarely any guarantee that *all* accidents are reported to the surgical stations. Some factories have first aid boxes in the workshops. Unless there is very strict supervision and a high standard of reporting, workers are likely to bandage small cuts and the like for themselves without troubling to report them, unless, as frequently happens, they are compelled to report with a septic wound a few days later. Furthermore, considerable difficulty is often experienced in getting details regarding the length of time that workers have been exposed to risk. Short time and holidays, as a rule, affect all the members of a homogeneous group equally; but records of lost time through sickness and the dates when workers are taken on and discharged are quite often not available. The discovery of the age and experience of the workers is sometimes a matter of considerable trouble; and finally, even if all accidents have been reported, it is not always an easy matter to find out exactly how many workers have had no accidents at all.

Many of these difficulties can be obviated, however, by a careful choice of the groups of workers to be examined, and statistical analysis has shown that some of the other difficulties are unimportant. For example, major and minor accidents have been found to correlate fairly highly in some cases (though this is a point which needs confirmation), so that if a record of either alone is available it will serve as an indication of the varying accident proneness of the workers.

At this point the foregoing remarks

may be summarized into a statement of our position at the beginning of a practical research into the nature of accident proneness. We have, as the sole criterion of our success in discovering the functions involved in proneness, a set of accident records which may have been inaccurately kept, which will include an unknown percentage of accidents not due to personal proneness, and which will have an unknown correlation with accident proneness itself. We have then to devise a series of psychologic tests, the ability to perform which must be related as closely as possible to accident incidence. When this has been done, if we succeed in finding a test for every psychologic and physiologic function that plays a part in accident proneness, and if further we combine these test scores, each correctly weighted, to form a single combined score which shall have the closest possible relationship to accident proneness, even then we can never hope to get anything approaching perfect correlation between the combined score and accident incidence. The reasons for this have already been pointed out: The accidents recorded do not themselves correlate perfectly with proneness, the records include an unknown proportion of accidents of the inevitable type, and the functions tested probably are not involved to the same degree in the individual's reactions to different accident situations.

Relationship between Tests, Accident Incidence, and Industrial Proficiency

We now come to a consideration of the actual tests to be applied in an attempt to elucidate this problem. Psychologic tests may be grouped under the two heads of general and specific. Now it has not yet been shown that there is a specific test or tests for accident proneness. The sustaining of an accident may be due to so many different causes that it is impossible to devise an analogous test for accidents in general in the same way that a test may be devised for proficiency in a particular repetitive task. In other words, although the individual has a certain proneness to accidents, it may be that he has no special ability for their avoidance. Hence, until the existence of a specific factor or factors in accident proneness has been established, we can only apply tests of a general nature. These tests may be for functions at any mental level from pure reflex to ideation; and if we consider the nature of the various types of accidents which occur, we shall find no reason for assuming that the functions involved in accident proneness all lie at some definite mental level. Rather, the reverse is true. In some cases the accident may be due to failure or slowness in making the simplest muscular reaction, in some to poorness of neuromuscular coordination of a more complex type, and in others to sheer lack of intelligence. Hence it is necessary to use a number of tests which together cover as large a mental field as possible.

The ideal battery of tests would include one for each function involved in accident proneness. The actual relation of each test to accident incidence could then be found and all the tests weighted together in their correct proportions to yield a single score

which would have the highest possible correlation with accident incidence. Theoretically, as we have already seen, the correlation between this ideal group of tests and accident proneness would still fall short of unity to a degree which we have no means of measuring. For practical purposes it is of course desirable to limit the actual number of tests used to as few as possible. Such tests should not correlate with one another, since high intercorrelation in this case would mean unnecessary overlapping.

It should perhaps be said here that the foregoing depends on the assumption that all the functions involved in accident proneness are directly measurable. However, there is no reason to assume that psychologic functions which cannot be measured at our present stage of knowledge do not also play their part in accident causation. Temperamental differences may be cited as an example of such nonmeasurable functions. Here we have yet another factor militating against success in measuring accident proneness, since we have no means of scoring nonmeasurable functions and combining such scores with those of functions which are directly measurable.

So far only the attempt to measure accident proneness with the practical object of reducing industrial accident incidence has been discussed. The avoidance of a large accident rate is, however, only one of the problems arising in industry. One of the most important of the other problems is how to obtain the maximum efficiency from the workers, and it will be seen at once that, as a matter of practical politics, no employer can be expected to choose operatives with a low degree of accident proneness if by so doing he gets less efficient workers. This suggests a further aspect of the research into accident proneness—namely: What is the connection between accident incidence and industrial proficiency? To answer this question it is necessary to discover the relation between the tests used for accident proneness and such records of industrial proficiency as are available. Theoretically it would seem likely that there is a fairly high connection between a high accident proneness and a low standard of efficiency, for the accident situation is also a situation occurring in the performance of an industrial task and a reaction to this situation which results in an accident cannot at the same time yield an efficient performance of that task. However, the question can by no means be fully answered in this way since the number of accidents sustained by even the most highly accident-prone worker is very small in comparison with the number of tasks he performs unscathed. The only way to solve this problem satisfactorily is to see if the tests which correlate with accident rate also correlate with industrial proficiency.

In face of all the difficulties which beset an investigation of this sort, it is evident that the greatest care in technic and accuracy in analysis must be exercised if the results are to have any real meaning and value. It is not, as a rule, possible in the biologic sciences to keep all factors constant except the one we desire to measure. Therefore, it is necessary to examine the results obtained from several different angles in order to make sure that we are actually measuring what we set out to measure. To apply

certain tests to a group of workers and correlate the scores obtained with the accident records of the subjects is by no means sufficient to yield a reliable answer to the problem. High correlation coefficients may in isolated instances arise quite by chance, and even if they are not spurious they may be due to a relation quite different from that which they appear to measure on first sight. This can be guarded against only by repeating the same tests on as many different groups of subjects as possible and by subjecting all the results obtained to statistical analysis by such methods as that of partial correlation. Correlation coefficients which consistently repeat themselves in a number of different groups may be regarded as fairly reliable, and even if low in actual numerical value are probably definite indications of real relationships. It is necessary to examine the data with the utmost care at each stage in the investigation. The frequency distributions of the scores made in the tests, the intercorrelations of the tests themselves, the relationship between the tests and such variables as the age and experience of the workers, and the time of day at which the tests were performed—all these points have to be carefully examined for each group tested, and their influence, if any, on the final results eliminated before we can decide what meaning to attach to the results obtained.

An investigation of the nature described above, made over a large enough field, is of value quite apart from the practical solution of the immediate problem that it may afford. It will add new facts to the body of scientific knowledge and may definitely advance psychologic theory. As was said at the beginning of this article, such a research involves a thorough investigation into the interconnection of various mental functions; and provided the work is of sufficient scope, light will be thrown not only on the nature of these functions themselves but also on their relationships with one another and their relative importance in different psychologic situations.

Our attitude toward the study of accident proneness is pragmatic: We are concerned with finding tests which will correlate with accident incidence, no matter what they actually measure. However, it is useful from the point of view of theory to name tests even though we cannot define the functions which they measure. Hence a set of intercorrelating tests which bear a definite relation to what is commonly called intelligence may conveniently be called intelligence tests. Similarly another set of tests may be called aesthetokinetic tests and be said to measure aesthetokinesis.[2] Such names, of course, throw no direct light on the nature of intelligence and aesthetokinesis, but the nomenclature is useful for purposes of reference and comparison. For example, if we make the statement that both intelligence and aesthetokinesis play a part in engineering ability, what we actually mean is that there is some factor involved in both engineering proficiency and the ability to do well in certain

[2] Three sensorimotor tests which were found to intercorrelate were called aesthetokinetic tests because their successful performance required accurate and rapid coordinated reaction to perceptual stimuli, although the form or perceptual setting of the tests differed considerably. They therefore form a special group of sensorimotor tests, distinguishable from sensorimotor tests in general (4) (5).

tests which we call intelligence and aesthetokinetic tests. This is a definite advance in psychologic theory; for, without knowing in the least what the function we have called aesthetokinesis really is, we can definitely say that it is a function which is measured by a certain set of sensorimotor tests and that it plays a part in engineering proficiency and in the avoidance of accidents. The relative importance of the part it plays in the two cases may be determined by correlation.

This article would not be complete without a brief account of the practical work which has already been done in this field. In the course of several years Mr. Farmer and I have given thirteen different tests to a large number of groups of workers, totaling over 1,800 subjects in all. Full details of the early part of this investigation have already been published by the Industrial Health Research Board (4) (5), and I shall just indicate here, in a few words, the scope of the experiment and the chief facts elicited.

Tests were given designed to measure intelligence—expressed through both a linguistic and a mechanical medium—sensorimotor ability of various degrees of complexity, physiologic characteristics such as physical strength and muscular control, and nervous stability. The subjects, who were for the most part engineering apprentices, were carefully chosen so that as many as possible of the factors such as age, experience, and exposure to risk were kept constant. These subjects fell into two groups, those who had suitable accident records and those who had suitable records of industrial proficiency. Unfortunately, no group possessed both sets of records; consequently direct comparison between the relations of the tests to accident incidence and to industrial proficiency was not possible. This comparison could, however, be made indirectly.

It was found that a group of sensorimotor tests, those designated as aesthetokinetic tests, correlated with accident incidence, and that when their scores were weighted together with the entrance examination scores of the subjects, a final correlation coefficient of 0.406 with the accident rate was obtained. The correlation coefficient between the entrance examination alone and accidents was 0.294.

In the course of analyzing these results an interesting fact was revealed. It was found that where the accident records extended over several years, the relation between aesthetokinesis and proneness to accidents became more marked as the period of exposure to risk increased. Thus, while the coefficient of the correlation between aesthetokinesis and accidents for only one year was 0.099, it increased in size when the accidents were recorded for periods of two and three years, and became 0.444 when the period of exposure was four years. It is evident that the longer the period over which accident records are taken, the nearer does the actual number of accidents sustained come to being a true measure of accident proneness. Also, if a test really measures accident proneness, it is obvious that the better the measure of proneness, the higher will be the correlation between the test scores and proneness. Hence it follows that the relation between the test scores and accident incidence should become more marked as the period of exposure

increases. Since this condition is satisfied in the case of the aesthetokinetic tests, within the limits of the data available in this experiment, we have yet more evidence from a different standpoint to show that the aesthetokinetic tests really do measure a factor in accident proneness.

On examining the relation between the tests and industrial proficiency, it was found that three groups of tests correlated significantly with proficiency. First, there was the group of aesthetokinetic tests; second, a group of intercorrelating intelligence tests; and third, a dynamometer test for physical strength. Since these three groups of tests did not correlate with one another, we were able, by taking a single score for each group, to obtain three nonintercorrelating measures of industrial proficiency. When these were weighted together with the entrance examination scores of the subjects, a final coefficient of 0.472 with proficiency was yielded, the entrance examination alone giving a coefficient of 0.388 with proficiency.

Representative tests from each of these three groups had been given to four groups of subjects with suitable accident records. For these subjects, the tests were weighted into a single score, using the same weights as in the case of the correlation with industrial proficiency; and the correlation coefficient between this new weighted score and accidents was 0.446, which should be compared with the coefficient of 0.294 between the entrance examination alone and accidents.

These correlation coefficients, though statistically significant, are in no case very large; but a number of reasons have already been given to show that the maximum coefficient of correlation that could possibly be obtained falls short of unity by an unknown, but probably considerable, amount.

Summarizing these results it will be seen that tests have been found which, when combined with the entrance examination of apprentices to a skilled trade, correlate more highly with accident proneness than does the entrance examination alone. Also, the same tests combined with the entrance examination correlate more highly with industrial proficiency than does the entrance examination alone. Therefore, if the entrance examination of these engineering apprentices were supplemented with certain of the psychologic tests, the candidates selected would on the whole be more proficient and less liable to have accidents than those selected by the present methods. The experiments are still in progress, and in the course of time it will be possible to publish a more extensive report in which the results of new tests for other psychologic functions, as well as the results of applying the same tests to further groups of subjects, will be given.

Conclusion

In conclusion I should like to emphasize the fact that although the study of accident causation is full of difficulties, as we have seen in the foregoing survey of the problem, yet certain definite advances have been made. That individuals differ in their proneness to accidents has been established beyond dispute, and it has further been shown that certain measurable psychologic functions play a definite part in this proneness. Moreover, imperfections in

the accident records militate against success in establishing the existence of a relation between any tests and accident incidence, so that any correlation that is found is not due to faulty data. Hence the results that have already been obtained must not be discounted by the fact that the data are imperfect; on the contrary, it may be said that if the data were free from error, the relation shown to exist between aesthetokinesis and accident proneness would be seen to be still closer than now appears.

Stress has been laid upon the extreme care and accuracy necessary to produce reliable results in an investigation of the sort described in this article. This, of course, only applies to the actual investigation itself. Once relations between certain psychologic functions and accident proneness have been definitely established, the tests measuring these functions may be used as a practical basis for selection, with the effect of lowering accident rate and increasing industrial proficiency.

The results presented in the foregoing are most encouraging and suggest that further research along similar lines may show what other functions are involved in accident proneness and may yield results that will be of great practical value.

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TOTAL HEAT DIFFERENCE AS A MEASURE OF WET KATA COOLING*

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IN 1922, O. W. Armspach and Margaret Ingels (1) proposed as the measure of wet kata cooling rate, at a given velocity, the difference between the total heat content of air saturated at the average temperature of the kata-thermometer and the total heat content of air saturated at the wet bulb temperature. This idea received little attention from other investigators until lately, when a number of correlations have been attempted with varying success. If total heat difference can be made the basis for calculating cooling rate, it should be possible to develop a formula involving total heat difference which can be shown to give the same results as the rational formula published in a recent issue of THIS JOURNAL (2).

The rational formula for the rate of cooling of a warm body at constant temperature, and covered with a film of moisture, was shown to be

$$n\,(T_k - T_w) = R\,(T_w - T_s) + C[(T_w - T^1) + 91\,(e_w - e^1)]$$

- n = Combined coefficient of conduction and radiation into the water film, from the body.
- T_k = Temperature of body, in degrees Fahrenheit.
- T_w = Temperature of surface of water film, in degrees Fahrenheit.
- T_s = Temperature of surrounding objects.
- R = Coefficient of radiation.
- C = Film coefficient of conduction from water to air.
- T^1 = Wet bulb temperature of air.
- e_w = Vapor pressure of water at temperature T_w, in inches of mercury.
- e^1 = Vapor pressure of water at wet bulb temperature, in inches of mercury.

W. H. Carrier (3) has shown that the total heat above 0°F. in 1 pound of air can be calculated from the formula

$$H = C_p\,T^1 + L^1\,W^1$$

- H = Total heat in British thermal units per pound of dry air.
- C_p = Specific heat of air at constant pressure (0.24 British thermal unit per pound).
- T^1 = Wet bulb temperature, in degrees Fahrenheit.
- I^1 = Latent heat at wet bulb temperature (British thermal units per pound).
- W^1 = Weight of vapor in pounds associated with 1 pound of dry air, assuming saturated condition, at the wet bulb temperature.

Consider 1 mol of dry air saturated with moisture. If the barometric pressure is P and the pressure of the vapor is e, the pressure of the air is $P - e$. With every mol of air there will be associated $\frac{e}{P - e}$ mols of vapor, and with every pound of air there will be associated

$$\frac{e}{P - e} \times \frac{18}{28.98} = 0.621\,\frac{e}{P - e}\ \text{lbs. of vapor}$$

*Received for publication April 28, 1930.

where 18 and 28.98 are the molecular weights of water and air.

Total heat may then be expressed for a given wet bulb temperature

$$H = C_p T^1 + 0.621 \frac{L^1 e^1}{P - e^1}$$

Consider now a warm body at constant temperature covered with a film of moisture. The difference between the total heat of air saturated at the temperature of the water surface and the total heat of air saturated at the wet bulb temperature is

$$C_p(T_w - T^1) + 0.621 \left(\frac{L_w e_w}{P - e_w} - \frac{L^1 e^1}{P - e^1} \right)$$

In the temperature range from 60° to 95° the latent heat may be considered constant at the average value of 1,048 British thermal units.[1] Since the vapor pressures are small compared with the barometric pressure, they may be neglected in the denominators without great error, and the total heat difference may be written

$$C_p (T_w - T^1) + 0.621 \frac{L}{P} (e_w - e^1)$$

If the barometric pressure is 29.92 inches and L is 1,048, the expression becomes

$$0.24 (T_w - T^1) + 21.8 (e_w - e^1)$$

Take 0.24 outside of the bracket, and we have

$$0.24 [(T_w - T^1) + 91 (e_w - e^1)]$$

This is the total heat difference, the total heats being computed by neglecting the vapor pressures in the expression P − e. Comparing this expression with the last term of the rational formula, we see that the rational formula could be written

$$n (T_k - T_w) = R (T_w - T_s) + \frac{C}{0.24} (H_w - H^1)$$

where H_w and H^1 are the total heats at the temperature of the water and at the wet bulb temperature, computed by the approximate method.

Accordingly the cooling effect of the air can be computed either by the expression

$$C [(T_w - T^1) + 91 (e_w - e^1)]$$

or by the expression

$$4.16 C (H_w - H^1)$$

If actual values, not approximate values for total heat are used, the expression should be

$$3.94 C (H_w - H^1)$$

This factor was determined empirically by using values of total heat and vapor pressure from the psychrometric chart of Dr. E. Vernon Hill (4). The following are some values from the chart:

Wet Bulb Temperature °F.	*Total Heat B.t.u./lb.*	*Vapor Pressure in. Hg*
95	61.01	1.645
80	42.64	1.022
70	33.51	0.732
60	26.18	0.517

For example, assume that the temperature of the water is 95° and the wet bulb temperature 70°. The cooling effect of the air, by the rational expression, is, then

$$C [(95 - 70) + 91 (1.645 - 0.732)] = 108.08 C$$

By the total heat expression, it is

$$3.94 C (61.01 - 33.51) = 108.35 C$$

[1] From the Steam Tables of Marks and Davis.

The agreement, when any wet bulb temperature in the range from 60° to 90° is used, is within 2 per cent.

The heat balance equation expressing the heat loss in a given environment is as follows, when the total heat difference is used.

$$n\,(T_k - T_w) = R\,(T_w - T_s) + 3.94\,C\,(H_w - H^1)$$

Summary

The effect of air at constant velocity on the rate of cooling of a warm body at constant temperature and covered with a film of moisture can be expressed closely as a function of the difference between the total heat of air saturated at the temperature assumed by the surface of the water film and the total heat of air saturated at the wet bulb temperature.

The total heat of air saturated at the temperature of the body is in no way involved.

The rate of cooling of the warm body in a given environment cannot be expressed as a function of total heat difference, because total heat difference has to do only with the effect of the air, while the total rate of cooling is influenced by radiation exchange with surrounding objects.

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THE INFECTIVITY OF SILICOTIC TUBERCULOSIS*

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DR. COLLIS, in "The Statistical Characteristics of Dust Phthisis," published in THIS JOURNAL in November, 1926, referred, at the end of his paper, to "one last statistical peculiarity . . . . , the certitude of which rests upon rather insecure data . . . "; *viz.*, the supposed low infectivity of silicotic tuberculosis. He then set forth the evidence upon which this belief was based. It will, I think, be generally agreed that the evidence is mainly inferential in character, and that more positive data are needed before the fact can be regarded as established.

The examination and observation of "contacts" to cases of tuberculosis is now one of the statutory duties of British Tuberculosis Medical Officers. Accurate data regarding the infectivity of tuberculosis should, in time, emerge as a result of their labors. As far as silicotic tuberculosis is concerned, a special opportunity for settling a very interesting problem awaits the Tuberculosis Officers who work in areas where industrial silicosis prevails.

Although the examination of contacts was not so prominent a part of the work of a Tuberculosis Officer in 1926 as it is today, it seemed worth endeavoring to see whether some figures bearing on the matter referred to by Dr. Collis would not emerge from an investigation of the health of the families of the few cases of silicotic tuberculosis which I had under my care in the gritstone districts of Derbyshire, despite the smallness of their numbers. With that object in view, the following investigation was made in the winter months of 1927 to 1928.

Up to 1927 I had met with twenty-four cases of silicosis with pulmonary tuberculosis, mainly among the Derbyshire gritstone masons.[1] In every case the diagnosis had been verified by the presence of tubercle bacilli in the sputum, and the majority of the cases had already ended fatally. Although the number of cases was small, I thought that it might be worth while to investigate the condition of the widows, wives, and children of these men, and to compare it with the condition of the widows, wives, and children of a similar number of men with non-silicotic open tuberculosis, from the same district and of the same age at the time when tuberculosis was first diagnosed.

* Received for publication Feb. 6, 1930.

[1] The distribution of these cases according to occupation was as follows:

Gritstone bench hand	13
Gritstone scappler	4
Gritstone quarryman	3
Potter	2
Silica and ganister brickmaker	1
Cutlery grinder	1

I found, however, that in the district under investigation married men over 45 years of age rarely developed tuberculosis except they were silicotic; consequently I had to go a little further afield and to include some cases from outside the gritstone area in order to make up the necessary twenty-four controls. Finally, when I had collected the controls, I found that their ages averaged 52.5 years as against 48.5 years in the silicotic group.

The results worked out as follows:

	Tuberculosis with Silicosis	*Tuberculosis without Silicosis*
Cases:		
Total no.	24	24
Average age on diagnosis	48.5	52.5
Wives and Widows:		
Total no.	19	20
No. developing tuberculosis	0	1
Percentage developing tuberculosis	0	5
Children Born:		
Total no.	91	68
No. developing tuberculosis	13	8
Percentage developing tuberculosis	14.3	11.8
Children under 14 on Date of Fathers' Diagnosis:		
Total no.	23	9
No. developing tuberculosis	11	3
Percentage developing tuberculosis	48	33.3

The general rate of incidence of conjugal tuberculosis has been the cause of much controversy, but is, I think, now generally accepted as averaging about 5.6 per cent. of noninfected persons who marry infected persons; and the investigations of the Tuberculosis Officers of Lancashire have given us monumental figures of the percentage of children of tuberculous fathers who develop the disease—figures which show a higher incidence of the disease than in the cases of the forty-eight fathers detailed above. As far as the meager figures which I collected can be relied on, they show that for approximately the same age group, the silicotic fathers were slightly more infectious to their children than the non-silicotic. And further, the figures suggest that the apparent low infectivity of silicotic tuberculosis is attributable to the late age at which the tuberculosis develops.

The British workman, in the silica industries as in others, marries young. By the time he has reached 48 years of age, most of his children have grown up and passed the most dangerous period of susceptibility to tuberculous infection. In the exceptional cases in which young children were found in the households of the silicotic fathers, the tuberculosis incidence rate was very high.

The total absence of conjugal infection among the wives and widows of the silicotic cases is on all fours with the oft-quoted observation of George Bauer—better known as Georgius Agricola—who wrote, in 1557, that women could be found in the mining communities of the Carpathian mountains, who had been widowed six times over by miners' phthisis. The work of this German on miners' phthisis, published nearly 400 years ago, suggests to us that we might pay a little more attention today to German work on silica and its biochemistry, if for no other reason than because its priority is so unassailable.

The figure of 5.6 per cent. men-

tioned for conjugal tuberculosis is an "average" figure, and therefore subject to the usual fallacies of averages. There is, for example, little doubt that the incidence rate among young married couples is higher than 5.6 per cent., and that in middle age it is lower. There are many other factors to be considered, as well as the ages of the infectious and the noninfected spouse. Child bearing, lactation, housing, nutrition, educational and social condition, all have their influence. But when, eventually, all the factors which determine the incidence of conjugal tuberculosis come to be included in one comprehensive equation, we must not be surprised to find that the variable for "silicosis" has dropped out.

Meanwhile, facts are needed. It is in the power of the Tuberculosis Officers working in silicotic areas to pile up these facts, little by little, from the families of silicotic patients who come under their observation.

J. I. H.
June, 1930

THE OCCURRENCE OF PULMONARY FIBROSIS AND OTHER PULMONARY AFFECTIONS IN ASBESTOS WORKERS

(Concluded)

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Morbid Anatomy

Professor M. J. Stewart of Leeds has kindly supplied me with a detailed description (dated March 28, 1929) of postmortem appearances of several cases. From this account the following points are extracted:

The gross morbid anatomy of this lesion, as seen in persons who have been at work for many years in the dusty atmosphere of an asbestos factory, consists in a widespread pulmonary fibrosis affecting especially the basal region of both lungs. There are extensive, densely fibrous adhesions throughout the pleural sacs, which may, indeed, become completely obliterated. In particular, the bases of the lungs are firmly adherent to the diaphragm, and between these two structures there is often a thick, homogeneous layer of fibrous tissue, similar in appearance and consistence to yellow fibrocartilage. As a result of these two processes—pulmonary fibrosis and pleural sac obliteration—bronchiectasis develops in the midst of the more grossly diseased tissue and may go on to the formation of multiple abscesses with smooth walls.

The distribution of the fibrosis, apart from the extensive basal lesions, is rather irregular, the peripheral, subpleural regions of the lungs being more affected than the central areas. In one case in which the patient had been away from work for four years (following nineteen years in the mill), the areas of dense fibrosis were extraordinarily sharply circumscribed. The nodular character of the lesion which is so striking a feature of silicosis in its earlier stages is not met with in this disease.

The totally fibrosed areas of lung show grayish-black mottling, owing to immobilization of carbon. This tissue is excessively dense and completely airless; but there is no evidence of calcification. Other portions of lung show varying grades of the same process, and even the least affected parts are definitely tougher than normal. In none of the cases was there any evidence of an active tuberculous lesion.

Histology

The essential lesion is a chronic interstitial fibrosis of the lung. In the earlier stages, there is fibroblastic proliferation in the alveolar walls, which become increasingly thickened in consequence; and there is catarrhal desquamation of the alveolar epithelium. At this stage of the disease peculiar golden-yellow "asbestosis bodies" are found in varying, usually considerable numbers, both in the alveolar spaces and in the thickness of their walls. Detached portions of bodies may often be seen engulfed by alveolar phagocytes, or a large unbroken body may be partially surrounded by these cells. In the more advanced stages of the disease, the fibrosis of the lung is complete. Alveoli can no longer be made out; but the asbestosis bodies still remain embedded in the fibrous tissue. Varying quantities of carbon are also present, chiefly in little masses as though it had been contained within phagocytes. Chronic bronchitic

changes are present and all grades of bronchiectasis may be seen, in cases of long standing, from simply bronchial and bronchiolar dilatation, with walls still more or less intact, to large spaces filled with pus in which the original bronchial walls have become converted more or less completely into fibrous and granulation tissue. Many of the smaller arteries show obliterative endarteritis.

Reversionary metamorphosis of alveolar epithelium is frequent, and such alveoli are often filled with albuminous fluid, in which clumps of "bodies" may be found.

Asbestos Dust in the Lungs

Dr. W. E. Cooke has supplied (under date of June 25, 1929) details of his researches on asbestos dust in the lungs, and on the constitution of the curious bodies, which have been freely used in the following notes:—

Sections of lung and the results of digestion of the lung with trypsin show an enormous amount of fine black granular dust, much of which is carbonaceous. In addition, there are two striking features. The first is the almost complete absence of the fine translucent spicules of fiber which make up a great proportion of asbestos dust in factories. The second feature is the presence of large fragments varying in length from 10 to 360 microns. They are found in fibrotic and necrotic areas, singly and in groups. The particles are so large—masses of them are seen in some sections—that they must have occluded small bronchi and resulted in fibrosis of the surrounding area.

Comparing these large particles in the lung with those found in asbestos dust, close resemblance in sizes, shapes, and colors is apparent. There are the same black, blue, brown, and translucent fragments. In fact it is easy to take such a single particle from the lung and immediately find its brother in a slide made from the dust.

Curious Bodies

In addition to the fine granular dust and larger fragments of asbestos, sections of the lungs show curious bodies. They are found in alveoli, bronchioles, fibrous and necrotic areas, and in phagocytes in sections of both lungs. If a portion of lung is teased and extracted with water, or digested with trypsin, or, as Professor Stewart pointed out (1928), if a smear is made from the cut surface of the fresh lung, these bodies are seen in myriads. The larger bodies measure from 20 to 100 microns or more in length, and are of a golden-brown color. They may have single clubbed ends or may appear as elongated dumb-bells; some are filamentous, while others suggest a series of disks.

Single coccal and spore-like forms, and aggregations of these, and streptococcal forms are not uncommon; the color varies in the smaller types from a very pale yellow to a yellowish-brown. The bodies do not stain with any of the aniline dyes, but in chrysotile workers, they give the Prussian blue reaction for iron in varying degrees of intensity. These curious bodies have been found in every autopsy in pulmonary asbestosis. It would be unprofitable to retrace all the steps which led down many by-lanes during the course of the work, but I will mention a few pertinent details of interest.

Professor Stewart suggested to me

that a better method than simple extraction of the lungs with water or saline would be to digest portions with trypsin. During this process it was found that if the specific gravity was kept at about 1,070, the black dust and larger fragments of asbestos, as well as the partially digested lung tissue, sank to the bottom of the tube. By decanting the apparently clear supernatant liquid, and by centrifugalizing, neutralizing, and washing the deposit, the curious bodies could be obtained in a pure state. This was done, and sufficient material for X-ray examination was obtained. The bodies were attached to a hair with gum and subjected to a seven-hour exposure by Professor Bragg's method. If the bodies were mineral, the X-ray film would have shown a definite translatable atomic pattern. The films, however, did not do so, and we were then able to exclude the suggestion that the bodies were altered asbestos fiber. The result definitely pointed to the greater proportion of their composition being of nonmineral origin.

The bulk of the curious bodies is soluble in strong acids and alkalies, and if the solution is observed under dark ground illumination, their bases are seen as extremely fine spicules some of which by transmitted light would probably be invisible. Under a dissecting microscope it is possible partially to fracture the larger bodies and to show a central fine core.

The greater portion of asbestos dust consists of slender translucent fibers. In sections and extracts of the lungs there is a remarkable paucity of these fine spicules. The end-results of digestion show the fine granular dust and the large black, blue, and brown particles and what appear to be pieces of quartz. Relatively few fine spicules are found; but curious bodies of all descriptions are present in enormous numbers.

All these facts lead us to imagine the bodies to consist of a central nucleus of asbestos spicules, upon which colloidal aggregate of blood proteins plus, possibly, soluble fractions of asbestos and, in the case of chrysotile workers, an iron salt have been adsorbed and molded by currents in the bronchi and the alveoli.

The method of formation would appear to be as follows: The fine spicules of asbestos cause, by mechanical action on the bronchioles and alveoli, either minute extravasations of whole blood, or serous exudates which envelop them. Solution of any soluble fraction of asbestos takes place. The total amount of asbestos that is soluble must be extremely small, as is proved by the X-ray pattern of the curious bodies, but in the case of chrysotile workers some solution is suggested by the free iron reaction. We must remember, however, that the Prussian blue reaction may be due to the iron of the hemoglobin. Any surface in contact with a colloidal solution may act as an adsorbent, and in the present case the fine spicules must be considered to do so. Interaction between the soluble fraction of chrysotile and plasma proteins takes place, syneresis occurs, and, with the loss of water, the adsorption is rendered irreversible. The adsorbent is permanently ensheathed with stable colloidal aggregates which become molded into the familiar

shapes by alveolar and bronchial currents.

Support for this idea is found in the fact that micro-organisms adsorb colloidal material in the presence of blood serum and colloidal asbestos. Staphylococci so treated appear as large round yellowish-brown disks; in the process their property of staining with aniline dyes is lost. The organisms coalesce and form masses; and I think it probable that some of the coccal and spore forms are similar organisms.

Finally, are the curious bodies diagnostic of pulmonary asbestosis? Asbestos is unique among minerals in being fibrous; and its dust, generated during manufacturing processes, is also unique. As can be imagined from the formation of the curious bodies, there is no reason why, given any silicosis, a fine spicule of mineral should not have colloidal matter deposited around it and become molded into a curious body. But as no other mineral dust is fibrous, this occurrence must be so rare as to be negligible from a diagnostic point of view. The conditions which, apparently, must obtain for the formation of curious bodies are the presence of plasma proteins and fine spicules soluble only with difficulty. These conditions are ideally found in asbestos workers; for this reason I believe that curious bodies, if found in any numbers, are pathognomonic of pulmonary asbestosis.—

W. E. Cooke and also Roodhouse Gloyne (20) have independently demonstrated the presence of a mineral core, evidently derived from asbestos fibers, in the curious bodies. The bodies have been found (Stewart and Haddow (21)) in the intrathoracic lymphatic glands; in material obtained by lung puncture during life, first suggested by S. A. Henry; in the sputa; and in smear preparations from the cut surface of the lung. In smear preparations they can be readily demonstrated.

The importance of these findings lies in the fact that the bodies have never been found, as yet, in any other human affection. That similar bodies may be found to occur in the lungs of workers exposed to other dusts, is quite possible, although they have not been found in silicosis, nor were they present, according to Simson (6), in the fibrosed lungs of a hematite miner.

They appear within a comparatively short period of time after exposure to asbestos dust; at the moment, their presence in the sputa, in the absence of clinical or radiologic evidence of pulmonary fibrosis, cannot be taken as indicating anything more than previous inhalation of asbestos dust. The presence of the bodies, however, in the sputa of persons with signs of a diffuse fibrosis, or their presence in numbers on postmortem examination of a fibrosed lung, has evident implications.

Professor Stewart, who is continuing his work on this subject, has devised the following method of examining sputum for asbestosis bodies:

Half an ounce or so of sputum is added to an equal quantity of undiluted antiformin. This is gently agitated until the sputum is completely dissolved, after which it is diluted with 2 or 3 ounces of water and allowed to stand in a large test tube for three or four hours. The bulk of the supernatant fluid having then been decanted, the remainder is centrifuged at a moderate speed for ten to fifteen minutes. The whole of the supernatant fluid is now poured off, and the deposit transferred to an albumin-

ized slide, by means of either a platinum loop or a pipette. After thorough drying on a hot plate and final fixation over a bunsen burner, the film is very gently washed in water, dried, and mounted in Canada balsam. After a little experience the asbestosis bodies are readily picked up with the low power, and their true nature is then confirmed with the $\frac{1}{6}$-inch or oil immersion lens. As a rule they are present in very small numbers, perhaps only one or two in a whole film. In the case of some of the older workers, however, numerous bodies, up to one or two per field in certain portions of the film, have been found. It is obviously necessary to cleanse thoroughly all glassware, etc., used in making these examinations; otherwise there is a risk of contamination of subsequent specimens.

The films can be treated with hydrochloric acid and ferrocyanide of potash to demonstrate the Prussian blue reaction given by the bodies.

Asbestosis an Occupational Hazard

Out of a total of 374 workers examined, 105, or 28.1 per cent., were found to be affected with fibrosis of the lungs, in greater or less degree. But when these figures are corrected by the exclusion of cases in which previous work in dusty occupations (*e.g.*, quarrying, coal mining) may have been the prime, or a contributory, factor in the development of the fibrosis, ninety-five, or 26.2 per cent., of 363 workers were found to be affected with pulmonary fibrosis due to the inhalation of asbestos dust, and a further twenty-one, or 5.8 per cent., showed precursive signs of this disease.

This percentage may well be compared with the corresponding figures found by Middleton (22) in his examination of metal grinders (46.9 per cent.) and by Sutherland and Bryson (23) (24) in their examinations of potters (39.9 per cent.) and of sandstone workers (58.1 per cent.).

The precise significance of the lower figure found in this inquiry is not easy to determine. At first sight it seems to indicate that asbestos dust is less potent as a cause of pulmonary fibrosis than are the dusts containing free silica. Other factors, however—such as the measure of exhaust ventilation, or other means of reducing the concentration of dust, in the several industries; differences in the average length of employment of the comparable groups in the samples examined in the respective inquiries; and the relative numbers employed in the more dusty and the less dusty processes—will affect the crude incidence rates expressed by these percentages.

From comparison of the amounts of dust evolved—determined visually and by means of dust counts—in dusty asbestos processes, uncontrolled by local exhaust ventilation, with obviously defective exhaust ventilation, and with comparatively good exhaust ventilation, there can be no doubt that this preventive measure, which has been applied in some degree for more than seventeen years, has been a positive factor in minimizing the production of fibrosis—probably in the direction of lengthening the period before the fibrosis becomes fully developed.

Consideration given to the distribution of the workers examined both according to length of employment, and according to age, together with the incidence rates of fibrosis in each case, indicates the outstanding importance of length of employment (and hence length of exposure to dust), and the negligible effects of age, on the production of fibrosis. Thus age groups 30 to

39 and 50 to 59 have incidence rates of 30 per cent. and 37.9 per cent., respectively—not a wide difference, since the groups include workers in various processes exposed to different concentrations of dust. Moreover, the average length of employment in these two groups, excluding the cases of fibrosis, is also similar, but considerably less than the average length for all the cases of fibrosis (13.5 years).

No special susceptibility to the development of fibrosis is shown by young persons, unless it is considered that the figure of 8.7 years, the average length of employment of the cases of fibrosis in age group 20 to 29, has been shortened by increased susceptibility at ages under 20. As, however, the average age of this group of cases of fibrosis is 26.7 years, and also because the establishment of fibrosis does not necessitate immediate retirement from work, this is largely discounted.

TABLE 5.—DISTRIBUTION OF WORKERS EXAMINED WHO HAD BEEN EMPLOYED FOR FIVE YEARS OR OVER, TOGETHER WITH CASES OF FIBROSIS, ACCORDING TO PROCESS IN WHICH WORKER WAS LONGEST EMPLOYED

PROCESS	NO. EXAMINED	CASES OF FIBROSIS		AVERAGE LENGTH OF EMPLOYMENT IN YRS.	
		No.	Per Cent. of Group	Group Less Cases of Fibrosis	Cases of Fibrosis
1. Crushing, opening, disintegrating, and mixing	21	9	42.9	8.9	10.9
2. Carding	39	16	41.0	9.1	13.2
3. Spinning, twisting, doubling, plaiting, etc.	87	12	13.8	10.1	18.7
4. Mattress making	15	7	46.7	7.3	13.0
5. Weaving and associated processes	88	40	45.5	10.0	12.7
a. Cloth weaving	28	21	75.0	9.5	14.1
b. Band weaving	21	4	19.0	10.9	13.5
c. Cloth and band and unclassified weavers	14	4	28.6	7.2	9.5
d. Warpers, beamers, loom tuners, charge hands, and others associated with weaving	25	11	44.0	11.2	10.8
6. Miscellaneous processes and remaining unclassified workers	24	11	45.8	8.8	13.8
Total	274	95	34.7	9.6	13.5

The effects of length of employment are such that after five years' exposure, the incidence rate of fibrosis mounts rapidly, and after ten years increases almost in geometric progression. Table 5 shows the distribution of 274 workers examined, and of those with fibrosis, according to the process

in which they were longest employed. This was a compromise necessitated by the common customs in the industry of having several different processes going on in the same room, and of workers transferring from one process to another. Since the intention was to obtain some measure of the *relative* incidence of fibrosis in workers in the various processes enumerated, and since no case of fibrosis clearly due to asbestos dust was found among eighty-nine workers examined, who had been employed for less than five years, inclusion of this group of workers would only have introduced a varying source of error.

The processes differ, some very materially, in the amount of dust which they cause, and consequently in the amount inhaled by the operators (30). The outstanding point brought out by Table 5 is the relatively low incidence rate of fibrosis in "spinners" (group 3), as compared with each of the other groups. They are corroborated by counts of the dust particles in samples of air, a number of which have been taken and are discussed under *Dust Risk*.

There is also some indication in these figures that not only are "spinners" less likely to develop fibrosis, but when they do, it takes longer to develop. Study of the radiograms suggests that in such cases (*i.e.*, in the less dusty processes) the resulting fibrosis is, for a considerable period, much more linear in its radiologic features, and shows less mottling. The history and clinical features in these cases, and the comparative dust counts, all contribute to the view that with comparatively low concentrations of dust, the resulting fibrosis is longer in developing and remains longer in the milder, and radiologically linear stage.

This view is evidently of considerable practical importance since it suggests that in such cases, the rate of accumulation of dust in the lung has not greatly exceeded the rate of elimination, the changes still being centered in the main lymphatic system.

If this hypothesis is accurate, it should follow that in order to prevent the full development of the disease, among asbestos workers, within an average working lifetime, it is necessary to reduce the concentration of dust in the air of the workrooms to a figure somewhat below that pertaining to spinning at the present time.

Fortunately, also, the spinning group is the largest individual group in this section of the industry—*e.g.*, out of over 400 workers employed by one firm, this group accounts for more than one-third.

Although all workers examined who have been included as spinners have been engaged in spinning, or in similar processes for convenience termed "spinning," for a longer time than on any other process, only a few have been employed solely on spinning, and in workrooms where no other, and more dusty processes, were being carried on.

These two factors, prior work in more dusty processes and present work in proximity to more dusty processes—especially the latter—have had, it is believed, some effect in raising the incidence rate of fibrosis for this group.

Dust Risk

In an effort to obtain additional data on the influence of concentration of dust in workrooms, a number of samples of air were taken and the dust con-

TABLE 6.—SUMMARY OF DETERMINATION OF DUST CONTENT OF ATMOSPHERE OF WORKROOMS AND OTHER PLACES

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENTAGE 2μ AND UNDER	PERCENTAGE 7.5μ AND UNDER
1. 1/2	Carding; passageway between cards	Yes	Yes	2,139	81.3	95.1
2. 1/5	Spinning	No	Yes	1,456	95.9	100.0
3. 1/3	Cloth weaving, dry	No	Yes	1,756	84.1	96.7
4. 1/7	Opening	Yes	Yes	2,384	80.9	93.7
5. 1/6	Sieving; shoveling fiber on to sieve	No	Yes	2,829	83.0	97.6
6. 1/8	Plaiting	No	No	1,687	99.0	99.0
7. 1/1	Office	...	...	901	98.8	100.0
8. 2/11	Office	...	...	1,480	98.7	100.0
9. 2/1	Cloth weaving	No	Yes	1,788	88.9	98.6
10. 2/14	Spinning	No	Yes	1,073	96.2	100.0
11. 2/10	Mattress making; filling	Yes	No	985	92.5	98.1
12. 2/8	Mattress making; beating, damp	Yes	No	970	85.9	95.8
13. 2/7	Mattress making; beating, dry	Yes	No	1,629	86.9	96.6
14. 2/9	Mattress making; after filling 2 mattresses	Yes	No	1,204	96.6	99.4
15. 3/1	Band weaving, wet	No	No	808	96.6	100.0
16. 3/4	Opening	Yes	No	2,313	77.7	96.6
17. 3/3	Carding; between cards	Yes	Yes	1,528	82.5	97.4
18. 3/5	Carding; feeding card	Yes	Yes	1,321	82.9	96.4
19. 3/2	Office	...	...	1,049	96.3	100.0
20. 4/1	Mattress making; filling	No	Yes	1,858	60.1	90.1
21. 4/1	Mattress making; filling basket	No	Yes	1,390	....	.....
22. 4/3	Emptying settling chamber	No	Yes	3,401	....	.....
23. 4/4	Same	No	Yes	4,711	....	.....
24. 4/5	Insulating sections; leveling fiber	No	Yes	1,838	....	.....
25. 4/5a	Carding; side of dirty card	Yes	No	1,402	....	.....
26. 4/6	Carding; end of clean card	Yes	No	1,092	....	.....
27. 4/7	Carding; feeding card	Yes	No	1,689	....	.....
28. 4/9	Cloth weaving, very slightly damp	No	Yes	3,033	....	.....
29. 4/10	Cloth weaving, damp	No	Yes	758	....	.....
30. 4/11	Braiding	No	Yes	506	....	.....
31. 4/12	Spinning	No	Yes	954	....	.....
32. 4/13	Office	...	...	689	....	.....
33. 5/1	Band weaving, dry	No	Yes	2,202	....	.....
34. 6/2	Cloth weaving, dry	Yes	No	885	....	.....
35. 6/3	Band weaving, dry	Yes	No	701	....	.....
36. 6/9	Opening	Yes	Yes	643	....	.....
37. 6/6	Carding; feeding card	Yes	Yes	562	....	.....

TABLE 6.—*Continued*

SAMPLE NO.	PROCESS AND SOURCE OF SAMPLE	LOCAL EXHAUST VENTILATION TO PROCESS OR NOT	OTHER PROCESSES IN SAME ROOM	NO. OF PARTICLES PER C.C.	PERCENTAGE 2μ AND UNDER	PERCENTAGE 7.5μ AND UNDER
38. 6/4	Carding; center of room	Yes	Yes	620	....	.....
39. 6/5	Carding; grinding rollers	Yes	Yes	540	....	.....
40. 6/7	Spinning	No	Yes	620	....	.....
41. 6/8	Office	...	...	666	....	.....
42. 7/2	Cloth weaving, dry	Yes	Yes	4,688	....	.....
43. 7/1	Opening; shoveling fiber into box	Yes	Yes	4,880	....	.....
44. 7/3	Opening	Yes	Yes	6,324	....	.....
45. 7/4	Band weaving, wet	No	Yes	5,353	....	.....
46. 7/5	Spinning	No	Yes	6,044	....	.....
47. 7/6	Carding; between cards	Yes	Yes	4,607	....	.....
48. 7/7	Office	...	...	3,413	....	.....
49. 8/2	Cloth weaving, dry	No	Yes	1,287	88.6	94.9
50. 8/3	Band weaving, dry	No	Yes	1,233	78.2	93.7
51. 8/5	Carding	Yes	Yes	1,195	67.2	94.9

tent was determined by means of Owens' jet apparatus. Here also the undetermined effect of dust from neighboring processes must be noted. Nevertheless some information of practical value was obtained.

Reference to Table 6 shows that the counts ranged from 506 to 6,324 particles per cubic centimeter in the air of workrooms, and from 666 to 3,413 per cubic centimeter in the air of offices associated with the factories. In some cases there was a smaller count in a workroom than in an office of the same factory; this was due either to there being much fewer smoke particles in the air of the workroom, or to adherence of the smoke particles to the inorganic dust in the workroom.

The character of the dust in the two cases is, however, entirely different. In the air of the office almost all the particles are black, or brownish, and amorphous, evidently smoke; in the air of the workroom, the majority of the particles are, just as clearly, crystalline particles of asbestos, together with, frequently, some cotton fibers and numbers of black and brownish particles, some adherent to asbestos spicules and some free. Most of the latter are iron containing and are derived from the asbestos, and there are relatively few smoke particles. Isolated golden-brown scales or plaques, also derived from the asbestos, are scattered about the field.

The asbestos fiber first fragments longitudinally, and apparently this process can go on indefinitely, since there is no ultimate fiber comparable to a vegetable fiber such as cotton. The dust collected in the Owens apparatus contains numbers of these very fine fibers—some about 0.5 micron in transverse diameter, many less—which have fragmented transversely. Thus spicules of very diverse length—up to

about 80 microns—but about 0.5 micron broad, are found in numbers on the sample, together with the black, brownish, and yellow particles already mentioned. The great majority of the spicules are found broken down into particles of the order of 2 microns and less, many being about 0.5 micron and more or less rounded. The proportion of elongated to rounded particles seems to vary in the different processes; for example, a greater proportion of elongated particles is found in the air near dry cloth weaving than near a spinning frame. Weaving damp reduces the proportion of elongated particles.

This variety of microscopic spicules, particles, and frequently some cotton fibers, found in the samples of dust collected in the Owens apparatus, results in a tendency to clumping on the cover slip, making counting, especially of the higher concentrations, a matter of extreme difficulty. Occasionally, also, part of the slot of the apparatus choked up with dust. The effect of the clumping is to reduce the counts of all except the low concentrations.

Furthermore, this apparatus is much influenced by local air eddies and currents. It takes a grab sample, which is accurate enough as an indicator of the local conditions at the moment of sampling, but does not give a picture of the average state of the atmosphere during a period of time, as does a gravimetric apparatus. Moreover, with high concentrations of dust, accurate counting becomes impossible.

The apparatus has, however, many advantages, including those of convenience, quickness in operation, and direct examination of the nature of the dust. By serial observations, too, temporary and local large increases in the dust content of the atmosphere—such as that produced by shoveling asbestos fiber into a sack—can be detected and estimated. This is of great importance since short, but repeated, exposure to massive concentrations of dust is agreed to be very harmful. Counts Nos. 5, 22, 23, and 43 in Table 6 are examples. Much useful work can be done by means of serial dust counts in various dusty processes in the same factory, and by comparison of the results.

The counts set out in Table 6 are derived from eight different factories, and it is a matter of interest to consider whether counts from different factories can be directly compared with one another. Strictly they cannot, since the content of smoke particles varies in each group. The difficulty might be obviated by counting only the particles derived from asbestos. The method has been applied successfully to counts of crystalline silica dusts; but it is not so easily applicable to counts of asbestos dust, which contains numbers of black and brownish iron containing particles, many of which are not quickly distinguishable from smoke and tarry particles.

To classify the processes accurately on the basis of amount of dust evolved, it is necessary to take serial samples at intervals corresponding to the details and complexity of the processes. With some straightforward processes, such as spinning, few samples will be necessary; but in others, such as mattress making, mixing, grading and opening—especially with old-fashioned plant—in which the evolution of dust rises and falls rapidly in a comparatively short space of time, many more observations are necessary throughout

a shift, to determine the average concentration of dust. Repeated controls are also required.

Comparative data show that the dustiest processes are opening (with old-fashioned teasers), sieving (with no local exhaust ventilation), and shoveling, or otherwise handling asbestos fiber. The heaviest counts of all were found in this group, in filling sacks, by hand, with disintegrated fiber in a settling chamber. The visual cloud of dust was intense, and irritating enough to provoke immediate coughing on the part of the observer. The clumping of the dust particles of the sample on the cover slip was very marked, with the result that the total dust count arrived at was undoubtedly too low. With counts of asbestos particles of this order, however, the precise count is of little practical importance, since the dust concentration is far beyond the safe limit.

Next in order is cloth weaving, dry and without the application of local exhaust. This is undoubtedly a dusty process, and although local exhaust reduces the count at the breathing level of the weaver, much dust still escapes into the workroom. Further counts are necessary to estimate this. Weaving cloth wet, not merely damp, reduces the dust count to a remarkable degree. The figure for band (*i.e.*, narrow) weaving, wet, is not accurate since it was raised by dust from a neighboring dry cloth loom; still the improvement due to wet methods is noticeable. One asbestos weaver, who was formerly a cotton weaver, and who has to wear glasses, stated that whereas he used to clean his glasses about three times a day when cotton weaving, he has to clean them about five times a day when asbestos tape weaving dry, and only once a day when weaving asbestos wet.

Next in order comes mattress making, without any precautions such as exhaust ventilation, or damping floors, cloth, and tables. Application of exhaust ventilation and damping reduce the count considerably, but it is believed that the figures are too low, since counts for some subsidiary processes such as buttoning, sewing, and cutting out are not available.

The figures for spinning, plaiting, and braiding are believed to be rather too high, owing to contamination of dust from neighboring and more dusty processes, and further investigation is required here.

A number of the samples taken with the Owens apparatus were examined to determine the size of the particles, and the percentages of particles 2 microns and under, and 7.5 microns and under, were ascertained. The 2-micron standard was retained for purposes of comparison, and because of its accepted importance in silicosis. The 7.5-micron figure was also adopted because of the pronounced acicular character of much of the asbestos dust, and because, representing the diameter of the human red blood corpuscle, it is a standard easily distinguishable, and may conceivably represent the limit size of particles which can be conveniently engulfed by phagocytic cells, which are somewhat larger. Among twenty counts from various processes (Table 6), in two, between 60 and 70 per cent. of the particles were of sizes 2 microns and under; in two, 70 to 80 per cent.; in ten, 80 to 90 per cent.; and in six, 90 per cent. and upward. When the counts were analyzed ac-

cording to the 7.5-micron standard, all twenty showed 90 per cent. or more of the particles to be of this size or less.

With regard to the effect on the lungs of different varieties of asbestos, no evidence was found to indicate that any one of its varieties is more potent in producing fibrosis than the others, other factors, such as concentration of dust, being equal. There is now no doubt, however, that both chrysotile and crocidolite will produce fibrosis; while the third, the comparatively newly discovered amosite, resembles crocidolite so closely in its chemical constitution, and in the characteristics of the dust, that there can be no reasonable doubt, also, with respect to it.

Length of Dust Exposure

No case of diffuse fibrosis clearly due to asbestos was discovered with under 5 years' employment. Three cases were found with 3, $3\frac{1}{2}$, and $4\frac{1}{2}$ years' work, respectively, who showed clinical signs of fibrosis. Definite confirmatory radiologic evidence was obtained in the first, definite but slighter radiologic changes in the second, and indefinite suggestive changes in the third. The previous occupation in the first case was flax weaving for 18 years; in the second, coal hewing for 16 years; and in the third, work in an iron foundry for 1 year. All these occupations are dusty; and in the last two there may be some exposure to free silica. Also increased silica content has been found in the lungs, after incineration, of flax dressers. For these reasons, these three cases could not be certainly ascribed to asbestos dust, although it was, probably, at least a contributory factor. Among the cases of fibrosis found, thirty-six had been employed in asbestos for periods ranging from 5 to 10 years; two of these cases, one employed for 9 years and the other for 7 years, show that a considerable degree of fibrosis may occur with less than 10 years' exposure.

F. W. Simson (6) examined the lungs of a guinea-pig which had been experimentally dusted by Mavrogordato for two hours a day on each of fifty days between February and April, 1925, and which died from other causes in December, 1927. He states that histologic sections showed a slight generalized fibrosis. This observer also examined portions of the lungs of two native asbestos mill workers; one had been employed for twelve months and had died from a miliary tuberculosis, and the other, employed for two years, had apparently never recovered from an attack of lobar pneumonia a year before death.

In commenting on the amount of fibrosis found, he states that "a comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal," and, again, that "the amount of fibrosis in two of the human cases . . . . was quite definite, and, if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present-day non-infective silicosis on the Rand."

Experiments by Professor Beattie, in 1912, demonstrated that the lungs of guinea-pigs exposed to asbestos dust for forty-three and sixty-seven hours showed "definite cellular proliferation, though not very extensive, and this is

certainly a preliminary stage in the production of fibrosis."

These pathologic and experimental findings suggest that the inhalation of asbestos dust rapidly causes some changes in the lungs. But no evidence was obtained that asbestos dust can produce an acute type of fibrosis comparable to that formerly noted in South Africa after repeated exposure to massive concentration of free silica, a few cases of which have occurred recently in Great Britain, causing death in from two to four years.

The amount of disablement produced by the development of pulmonary fibrosis in these workers is surprisingly slight for a number of years, even more so than is generally the case in silicosis. The nature of the work, however, in the majority of the processes does not involve much physical exertion—in fact, in this respect it is "light work." The affected person may, and often does, continue at work —with occasional intermissions, latterly, due to exacerbations of bronchitis—until the condition is advanced, although he suffers increasing inconvenience from shortness of breath on the slightest exertion.

Usually these cases cease work a year or more before death, but sometimes a terminal bronchopneumonia, or other acute infection, commences while they are still at work, and there is no long period of invalidism.

Fatalities

Particulars of eight deaths have been collected, and are summarized in Table 7. In six of these, advanced pulmonary fibrosis was the primary cause of death. In the remaining two cases, pulmonary tuberculosis was a contributory factor. In six of the eight cases the diagnosis was verified by postmortem examination; in the other two cases, confirmatory radiographic evidence was obtained.

Information has recently been obtained in regard to three other cases, in which death occurred in 1929. The details of one have been published by Wood and Page (25). Postmortem examinations have been made in all three cases, and in all, the presence of pulmonary asbestosis, without tuberculosis, was verified. In one case, the pulmonary fibrosis was the primary cause of death; in one, information as to the extent of the fibrosis has not yet been obtained; and in the third case, a lobar pneumonia was superimposed on the fibrosis.

It is not suggested that these few fatalities, in which the cause of death has been verified by strict inquiry, are any criterion of the true effect of the disease on the mortality rates of asbestos workers. Others are known to have occurred in which the existence of the asbestos fibrosis has been determined in life, but no postmortem examination has been possible.

Difficulties in Diagnosis

Many factors have contributed to impede both the recognition of individual cases of this disease, and the establishment of asbestos dust as the determining cause. These factors are consequent, partly on the nature of the disease, partly on the nature of the dust, and partly on the industry itself, its rapid growth and internal conditions. Difficulties in diagnosing the disease, its points of resemblance, in its latest stages, to fibroid tuberculosis, and the liability of those affected to be

TABLE 7.—PARTICULARS OF FATAL CASES

NO.	CASE	DATE OF DEATH[1]	SEX	AGE	YRS. EMPLOYED	MAIN PROCESS	CONDITION OF LUNGS	POST-MORTEM EXAMINATION	INQUEST	REMARKS
1.	Montague Murray case	6/13/1900	M	34	14	Card room 10 yrs.	Advanced pulmonary fibrosis; no tuberculosis	Yes	No	.....
2.	Cooke's case	3/14/24	F	33	18[2]	Spinner latterly	Extensive pulmonary fibrosis and tuberculosis	Yes	No	Exposed to dust from other processes.
3.	H. S.	1927	F	48	24[2]	Mattress maker	Pulmonary fibrosis and tuberculosis	No	No	Two radiograms confirm diagnosis of pulmonary fibrosis.
4.	M. S.	3/ 7/27	F	34	20+	Mattress maker	Chronic pulmonary fibrosis; no tuberculosis	Yes	No	.....
5.	Grieve's case (W. L.)	3/24/28	M	34	13½	Mattress beater	Advanced pulmonary fibrosis; terminal bronchopneumonia; no tuberculosis	Yes	Yes	.....
6.	M. M.	8/25/28	F	34	15–16	Mattress maker	Advanced pulmonary fibrosis; no tuberculosis	Yes	Yes	Worked for less than the period stated, as a mattress maker.
7.	J. C.	10/13/28	M	36	18	Weaving 1 yr.; mattress dept. 7 yrs. and 2 mos.	Advanced pulmonary fibrosis; no tuberculosis	No	No	Two radiograms confirm diagnosis.
8.	L. H.	2/ –/29	F	41	19	Mattress maker	Advanced pulmonary fibrosis; no tuberculosis	Yes	Yes	.....

[1] Arrangement of dates follows the American custom—*i.e.*, month, day of month, year.
[2] Estimated.

carried off by some intercurrent disease—which alone is noted on the death certificate—are all of importance.

A concrete example encountered during the inquiry will illustrate one of these points. J. C., Case No. 7 in Table 7, was known by his own doctor and by the Chief Tuberculosis Officer of the town to be affected with advanced fibrosis of the lungs. Four or five months before his death it was thought that a change of air would be of advantage to him. Through the generosity of the firm with which he had been employed, he was transferred from the town to a farm in the country, where he died. The death certificate, given locally, stated that pulmonary tuberculosis was the cause of death. In another case chronic bronchitis was given as the cause of death.

In the less advanced stages of the disease, the symptoms are so unobtrusive that the worker rarely consults his doctor. In the later stages, bronchitis, or the supervention of acute bronchopneumonia, pulmonary tuberculosis, or other acute infection, all so much more common, masks the underlying fibrosis; and the terminal condition is noted as the cause of death.

In the second place, the silicate dusts —of which asbestos dust is one—representing silica in the combined form as opposed to free silica, have naturally been overshadowed by the silica dusts, since they do not produce the picture of silicosis and have not been shown to be associated with an increased mortality from pulmonary tuberculosis.

Moreover, although the asbestos industry has very rapidly expanded, it is still comparatively small, and scattered over the country. A large proportion of the workers have been employed in asbestos only for comparatively short periods, and of the processes considered so far, the less dusty spinning group is at the same time the largest. Thus only now the existence of a health risk in the industry is beginning to be recognized.

Does Asbestos Dust Predispose to Other Pulmonary Diseases?

The important question as to whether asbestos workers show an increased liability to other diseases of the lungs, such as pneumonia, pleurisy, and pulmonary tuberculosis—especially the latter—requires further investigation.

A history of pleurisy was given by ten workers, in eight since commencing work in asbestos; in addition, one worker had a slight pleural effusion at the time of examination, and one showed signs of old pleurisy, but no history was obtainable. Of the two with attacks prior to work in asbestos, one had normal lungs, and the other showed signs of a bronchiectasis. Of the eight giving histories of pleurisy, three showed signs of fibrosis, four showed signs of the old pleurisy only (one being due to a gunshot wound during the War), and the eighth showed signs of an old inactive tuberculous lesion of the right upper lobe.

A history of pneumonia was obtained in sixteen workers—in ten prior to commencing work in asbestos, and in six since. Of these six, two showed signs of a thickened pleura only, one showed merely enlarged lung roots, two had diffuse fibrosis, and in one the lungs were normal. Of the remaining ten, one had a thickened pleura, two bronchiectasis, two diffuse fibrosis, one

commencing fibrosis, and in four the lungs were normal.

With regard to evidence of bronchitis, and bronchial and pulmonary catarrh, fifty-eight of the 363 workers (16 per cent.) showed signs of one or other of these conditions, all being quite mild. Of these, thirty-one also showed signs of diffuse fibrosis, representing 32.6 per cent. of the fibrotic group, and eight were in the prefibrotic stage (38.1 per cent. of that group). The fact that the clinical examinations were conducted during the warmer months of the year, no doubt operated to reduce the incidence of these complaints. Under the circumstances, the preponderance among the cases of fibrosis is the more noteworthy, as indicating persistent irritation resulting from the dust.

Of other diseases, eight gave histories of rheumatic fever, and of these, two had definite valvular disease of the heart. In addition, valvular disease of the heart was noted in one worker, but no history of rheumatic fever was obtained.

With regard to pulmonary tuberculosis, so definitely an added risk in silicosis, this disease group has been drawn up to include all workers presenting signs indicating a past or present pulmonary tuberculous infection, but excluding cases of old inactive hilar tuberculosis. Under this heading are included not only workers with definite clinical signs of active tuberculous lung infections, or of old inactive lesions, equally definite, but also those workers showing signs of old apical collapse, usually attributed to a past tuberculous infection.

Signs of the latter are not uncommonly found in quite healthy persons, and are of no importance unless it is shown that the inhalation of asbestos dust is associated with pulmonary tuberculosis to an increased degree, when such cases, as indicating a group of workers with a latent infection, would assume a greater significance. No close association is apparent, however, since only thirty-seven, or 9.9 per cent. of the 374 examined, showed evidence of this disease, excluding cases of old inactive hilar tuberculosis, even when those showing the minor changes mentioned above are included. In only four was there a family history of pulmonary tuberculosis; and three out of four active cases belonged to this group. Thirty-three cases were inactive, of which twenty-one presented no evidence of dust fibrosis, and twelve presented evidence.

Out of the 374 persons examined, fourteen, or 3.7 per cent., gave a family history of tuberculosis. Evidence of active pulmonary tuberculosis was present in three; in two of these three the source of infection appears to have been a near relative.

Thus, no outstanding susceptibility to pulmonary tuberculosis was disclosed, either among asbestos workers as a class, or among those with fibrosis, considering the frequency of old healed apical lesions among the general population.

We have, however, by no means disposed of the question. The supervention of a tuberculous infection on a lung already the subject of fibrosis produces an increase in symptoms, previously unnoticed or disregarded, and causes the worker to seek his doctor's advice. He is then appropriately advised to give up his dusty employment, migrates from the industry, and

may or may not accept sanatorium treatment. Thus there tends to be a drift of such cases from the fibrosis producing industry.

During the course of the inquiry, information was obtained of a number of persons, previously employed in asbestos, who were either at home or in sanatoriums, suffering from chest complaints. Where an examination of workers is confined to those at work, a certain number of advanced cases of fibrosis, and a certain number of cases of pulmonary tuberculosis, with or without an asbestos fibrosis, in persons who have either given up work, or who are off work temporarily, will be missed.

It is necessary, therefore, to leave this question of increased susceptibility to pulmonary tuberculosis in abeyance, pending further investigation.

With respect to any increased susceptibility to the supervention of other lung diseases in workers with an asbestos fibrosis, the data obtained were insufficient to lead to any conclusion, except as to bronchial catarrh. There are indications, however, that when pneumonia or bronchopneumonia supervenes on a fibrotic lung, the prognosis is grave.

Asbestosis and Silicosis Contrasted

In view of the relationship between the asbestos fibrosis and silicosis, since they are both varieties of fibrosis of the lungs caused by the inhalation of dusts, it seems desirable to summarize shortly the main points of similarity and dissimilarity between the two diseases, so far as has been ascertained.

1. Inhalation of asbestos dust, under favorable conditions of concentration of dust and length of exposure, will produce a serious and even fatal degree of pulmonary fibrosis.

2. The type of fibrosis produced, however, exhibits a number of divergences from that caused by the inhalation of free silica dust, and the outcome of these variations is not yet apparent.

3. The divergences noted so far are exhibited in the radiologic picture, and on postmortem examination of the affected lungs, both in the gross morbid anatomy and in microscopic sections.

4. In the latter case the appearances of the asbestos fibrosis seem to be sufficiently distinctive not to permit of confusion between it and silicosis.

5. The radiologic appearances of the asbestos type of fibrosis, even in the more developed stage, are more delicate, softer, and more diffuse than the silicotic fibrosis.

6. It follows, therefore, that an opinion as to the degree and intensity of an asbestos fibrosis, based on a comparison of the radiologic changes with those shown in standard silicosis films, will be an underestimate.

7. In the absence of a full medical and industrial history, possibilities of confusion of the two types of fibrosis will arise in the interpretation of radiograms.

8. There is evidence that other inorganic dusts, containing no free silica, may be productive of this fine type of fibrosis in varying degree.

Summary

1. There is a definite risk of the development of a diffuse pulmonary fibrosis in persons exposed to the inhalation of asbestos dust.

2. This risk varies directly as the length of employment (and hence

length of exposure to dust), and is unaffected by the age of the worker.

3. There is a considerable difference between the least dusty and the most dusty processes investigated, and there is a corresponding variation in the relative risk of the development of fibrosis in the workers in these processes.

4. With continued exposure to high concentrations of dust, the disease may be fully developed in seven to nine years, and may cause death after about thirteen years' exposure, exceptionally in a shorter period.

5. Fibrosis as it occurs in asbestos workers differs considerably from the disease as it occurs in workers exposed to free crystalline silica dust; it differs in the radiologic picture, and also on postmortem examination of the affected lungs.

6. The more dusty processes are those involving the preparation and manipulation of the raw asbestos (other than crushing), dry cloth weaving, and mattress making.

7. The less dusty processes are spinning and similar processes, and processes, other than dry weaving, involving the manipulation of the spun yarn.

8. Further investigation is required to determine whether there is any increased susceptibility to the supervention of pulmonary tuberculosis associated with the development of the asbestos fibrosis.

Much generous assistance has been received from many sources during the course of this inquiry, and for this acknowledgment is gratefully tendered.

In addition to those already referred to, the writer is beholden especially to Dr. S. A. Henry, who did much to facilitate the investigation, particularly by his detailed reports on various relevant matters, and by much organization and liaison work; to many others of his colleagues in the different districts, to a number of directors and officials of various firms, foremen, and employees; to Dr. W. E. Cooke of Wigan and Professor M. J. Stewart of Leeds for pathologic material and for much information as to the progress and results of their own researches into the gross anatomic and the histologic features of the asbestos fibrosis, and the constitution and location of the curious bodies; to Dr. MacGregor, Medical Officer of Health for Glasgow, for various clinical and radiologic facilities, and for his ever-present readiness to lend the aid of his Department, and especially to Dr. H. E. Seiler of his staff; to Dr. J. C. Robertson for the loan of a radiogram, together with detailed clinical notes of one patient and other valuable information; to Dr. W. H. Bateman for the loan of a radiogram, for some clinical histories, for obtaining and examining several specimens of sputum, and for other aid; and to Dr. J. Rennie, Chief Tuberculosis Officer at Sheffield, for a series of radiograms showing various stages of silicosis.

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BOOK REVIEW

RECENT ADVANCES IN PREVENTIVE MEDICINE. By *J. F. C. Haslam*, M.C., M.D., M.R.C.P., D.P.H., Assistant Director, Bureau of Hygiene and Tropical Diseases; Director of Library Services, London School of Hygiene and Tropical Medicine; formerly Government Medical Officer of Health, British Guiana. With a chapter on the Vitamins. By *S. J. Cowell*, M.A., M.B., M.R.C.P., Professor of Dietetics in the University of London. Cloth. Pp. 328 with index and illustrations. Philadelphia, Pa.: P. Blakiston's Son & Co. Inc.; London: J. & A. Churchill, 1930.

In these days the flow of new work is too great to permit anyone to keep abreast of literature in more than a small field; hence the value of the book which Dr. Haslam has just produced. In it he deals with various sides of preventive medicine: with new thoughts on eugenics; with investigations into maternal mortality; with wastage of young life and research carried out into the troubles of childhood and of school time. New work upon milk in relation to both its value and its capacity for carrying disease is fully discussed. A chapter which brings together present knowledge on vitamins, contributed by S. J. Cowell, is one of the best in the book, wherein, however, the level throughout is high. Our readers will find particular delight in the section devoted to atmospheric conditions, in which is admirably summarized recent work done both in Great Britain and in America relating to effective temperatures and the value of the katathermometer. The author shows that what at first appeared to be divergent views are in the long run only the same matters seen from different viewpoints. Another section is devoted to hygiene in industry, and herein investigations into the influence of the personal factor upon the cause of industrial accidents are admirably set forth; they show that this aspect has been largely neglected, even though it accounts for the lion's share of this preventable form of mortality and morbidity. Investigations into telegraphists' cramp are described, and the thought which has emerged with regard to the effect of psychologic influences upon occupational neurosis is stressed. Pulmonary asbestosis receives reference; it is an example of the harm which dust can do as apart from the dust of silica with its characteristic association with tuberculosis. Not the least interesting part of this section deals with an inquiry into sickness in the printing industry. From it the reader will appreciate the great difficulties which are associated with attempts to elucidate from sickness data the relation between environment and illness.

The book closes with a discussion of active immunization with special reference to the common cold, a matter which is of such constant interest to all, but particularly to communities which gather together under industrial conditions, and suffer therefrom much inconvenience and short time absenteeism. The book is one which can be recommended.—*Edgar L. Collis.*